Int. J Med. Pharm. Res. (P-ISSN: 2958-3675 | E-ISSN: 2958-3683)
Available on: https://ijmpr.in/

International Journal
of Medical and Pharmaceutical Research

CASE REPORT **OPEN ACCESS**

Bell's Palsy: A Case Report

Dr. Sanjana Velayudhan[1]*, Dr. Greeshma, K[1]

[1]Postgraduate 3rd Year, Department of ENT, The Oxford Medical College and Research Centre, QQP4+WXM, Yadavanahalli, Attibele, Bengaluru - Chennai Hwy, Rajajinagar 3rd Block, Anekal, Karnataka 562107, India

OPEN ACCESS

***Corresponding Author**
Dr. Sanjana Velayudhan

Postgraduate 3rd Year, Department of ENT, The Oxford Medical College and Research Centre, QQP4+WXM, Yadavanahalli, Attibele, Bengaluru - Chennai Hwy, Rajajinagar 3rd Block, Anekal, Karnataka 562107, India

Received: 12-10-2024
Accepted: 14-12-2024
Available online: 19-12-2024

A B S T R A C T

Bell's palsy represents the most common peripheral facial nerve paralysis, accounting for approximately 70% of all peripheral facial palsies. This idiopathic condition results in partial or complete weakness of facial expression muscles, accompanied by potential alterations in taste, hyperacusis, and changes in lacrimation and salivation. We present a case of a 45-year-old female daily worker who presented with a three-week history of facial weakness, difficulty drinking, drooling, headache, and right jaw pain. The patient had a confirmed history of HSV infection. Comprehensive clinical examination and electrodiagnostic testing revealed 50% facial nerve damage. The patient was successfully managed with a combination of antiviral therapy, corticosteroids, and supportive care, including ophthalmological intervention and physiotherapy. This case highlights the importance of prompt recognition and appropriate management of Bell's palsy, emphasizing the value of a multidisciplinary approach combining pharmacological intervention with appropriate supportive care and rehabilitation.

INTRODUCTION

Bell's palsy, first described by Sir Charles Bell in 1821, represents an acute peripheral facial nerve paralysis that typically presents with unilateral facial weakness [1]. This condition accounts for approximately 60-75% of all cases of acute facial paralysis, with an annual incidence of 15-30 cases per 100,000 population [2]. The facial nerve's complex anatomical course through the temporal bone makes it susceptible to compression and injury at various points, leading to the characteristic clinical presentation [3]. The etiopathogenesis remains a subject of ongoing research, although viral infection, particularly herpes simplex virus (HSV), has emerged as a leading causative factor [4]. Recent studies have demonstrated that viral-induced inflammation leads to compression of the facial nerve within the fallopian canal, resulting in edema, ischemia, and potential demyelination [5]. Additional viruses implicated in the pathogenesis include varicella-zoster virus, Epstein-Barr virus, cytomegalovirus, and human immunodeficiency virus [6]. Current understanding suggests that inflammation within the confined space of the facial canal leads to mechanical compression of the nerve, resulting in temporary or permanent damage to nerve fibers. This mechanical compression theory is supported by the observation that surgical decompression in select cases can improve outcomes [7].

Case Presentation

A 45-year-old female employed as a daily worker presented to our outpatient department with a three-week history of progressive right-sided facial weakness. The patient initially noticed difficulty in drinking water and excessive

drooling from the right side of her mouth, accompanied by persistent headache and right jaw pain. Her medical history was significant for a documented HSV infection, confirmed through previous serological testing. The patient denied any recent history of fever, trauma, or similar episodes in the past. Physical examination revealed marked facial asymmetry with complete right-sided facial weakness involving both upper and lower facial muscles. The patient demonstrated complete inability to close the right eye, with a positive Bell's phenomenon. Forehead wrinkling was absent on the affected side, and she could not smile symmetrically or puff out her right cheek. Otoscopic examination showed normal tympanic membranes bilaterally with no evidence of vesicles or other pathology in the external auditory canal. No lymphadenopathy or facial tenderness was noted, and the remainder of the cranial nerve examination was unremarkable.

Electrodiagnostic testing was performed two weeks after symptom onset. Electroneuronography demonstrated a 50% reduction in compound muscle action potential amplitude on the affected side compared to the healthy side, indicating significant axonal degeneration. Electromyography showed features consistent with acute denervation in the affected facial muscles. Complete blood count and metabolic panel were within normal limits, HSV serology was positive for HSV-1 IgG, and computed tomography of the brain showed no evidence of structural lesions or masses. Lyme disease serology was negative.

Based on the clinical presentation and diagnostic findings, we initiated a comprehensive treatment plan. The patient received acyclovir 800 mg five times daily for 7 days, along with prednisolone 60 mg daily for 5 days with gradual tapering over the subsequent 5 days. Vitamin B complex supplementation was also provided. Eye care included regular application of artificial tears during the day and lubricating ointment at night, with eye patch protection during sleep. Physical therapy was initiated, incorporating facial exercises, massage, and electrical stimulation therapy.

The patient underwent weekly monitoring for the first month, followed by biweekly visits for two months. By week 6, significant improvement was noted in eye closure, facial movement, and oral competence. Drooling had substantially decreased, and the patient reported improved comfort in social situations. Further follow-up was scheduled every 6 weeks for one year to monitor long-term recovery and detect any potential complications.

DISCUSSION

This case illustrates several important aspects of Bell's palsy management in clinical practice. The presence of confirmed HSV infection in our patient supports the viral etiology hypothesis, which has been extensively documented in the literature [8]. While the exact pathophysiological mechanism remains under investigation, the theory of HSV reactivation causing inflammation and subsequent nerve compression has gained significant support through molecular and clinical studies. The facial nerve's anatomical course through the narrow fallopian canal makes it particularly susceptible to compression effects from inflammatory edema, explaining the rapid onset of symptoms often observed in Bell's palsy [9].

The timing of presentation and initiation of treatment represent crucial factors in determining outcomes. Our patient's presentation three weeks after symptom onset was relatively late, as current evidence suggests that treatment initiated within 72 hours of symptom onset yields the best results. Despite this delay, the patient showed favorable response to therapy, possibly due to the moderate degree of nerve degeneration (50%) revealed by electrodiagnostic testing. This finding aligns with previous studies indicating that degeneration greater than 90% generally portends a poorer prognosis [10].

The decision to implement combination therapy with corticosteroids and antivirals was based on current clinical guidelines and the patient's confirmed HSV status. Recent meta-analyses support this approach, particularly in cases with severe paralysis or confirmed HSV infection [11]. Prednisolone acts by reducing inflammation and edema within the fallopian canal, while acyclovir addresses the underlying viral etiology. The gradual tapering of steroids helps prevent rebound inflammation while allowing time for natural recovery processes to begin.

Our patient's treatment protocol incorporated comprehensive eye care, which proves crucial in preventing corneal complications. The inability to achieve complete eye closure (lagophthalmos) poses a significant risk for exposure keratitis, emphasizing the importance of regular lubrication and nighttime protection. This aspect of management often requires close collaboration with ophthalmology services to monitor corneal integrity and adjust treatment as needed [12].

The role of physical therapy in Bell's palsy recovery remains an area of active research. While some studies suggest modest benefits from facial exercises and electrical stimulation, others emphasize the importance of preventing synkinesis through controlled, graduated facial movements. Our patient's positive response to rehabilitation supports the inclusion of physical therapy in the comprehensive management plan, though optimal timing and intensity of exercises require further investigation [13].

Psychosocial aspects of facial paralysis deserve special attention in the management protocol. The impact on social interaction, professional life, and emotional well-being can be substantial. Regular follow-up visits provide opportunities not only for monitoring physical recovery but also for addressing psychological concerns and providing reassurance. The importance of this supportive care cannot be overstated, as patient anxiety about permanent disfigurement can significantly affect quality of life during the recovery period [14].

CONCLUSION

This case underscores the importance of a systematic approach to diagnosing and managing Bell's palsy. Despite presenting later than the optimal treatment window, our patient achieved favorable outcomes through a combination of medical therapy and supportive care. The successful resolution of symptoms highlights the value of multimodal treatment incorporating antivirals, corticosteroids, appropriate eye care, and physical therapy. Regular follow-up allowed for monitoring of recovery and timely intervention for potential complications. Our experience supports current treatment guidelines while emphasizing the need for individualized care based on clinical presentation and progression. Future research directions should focus on optimizing treatment protocols and identifying reliable prognostic indicators to guide therapeutic decisions.

REFERENCES

1. Peitersen, E. (2002). Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol, 122*(4), 4-30.
2. Holland, N. J., & Weiner, G. M. (2004). Recent developments in Bell's palsy. *BMJ, 329*(7465), 553-557.
3. Baugh, R. F., Basura, G. J., Ishii, L. E., Schwartz, S. R., Drumheller, C. M., Burkholder, R., ... & Vaughan, W. (2013). Clinical practice guideline: Bell's palsy. *Otolaryngology–Head and Neck Surgery, 149*(3_suppl), S1-S27.
4. Kennedy, P. G. (2010). Herpes simplex virus type 1 and Bell's palsy-a current assessment of the controversy. *J Neurovirol, 16*(1), 1-5.
5. Lockhart, P., Daly, F., Pitkethly, M., Comerford, N., & Sullivan, F. (2009). Antiviral treatment for Bell's palsy (idiopathic facial paralysis). *Cochrane database of systematic reviews*, (4), CD001869.
6. Gilden, D. H. (2004). Clinical practice. Bell's Palsy. *N Engl J Med, 351*(13), 1323-1331.
7. Gronseth, G. S., &Paduga, R. (2012). Evidence-based guideline update: Steroids and antivirals for Bell palsy. *Neurology, 79*(22), 2209-2213.
8. Sullivan, F. M., Swan, I. R., Donnan, P. T., Morrison, J. M., Smith, B. H., McKinstry, B., ... & Daly, F. (2007). Early treatment with prednisolone or acyclovir in Bell's palsy. *New England Journal of Medicine, 357*(16), 1598-1607.
9. Eviston, T. J., Croxson, G. R., Kennedy, P. G., Hadlock, T., & Krishnan, A. V. (2015). Bell's palsy: aetiology, clinical features and multidisciplinary care. *Journal of Neurology, Neurosurgery & Psychiatry*, *86*(12), 1356-1361.
10. Gagyor, I., Madhok, V. B., & Daly, F. (2015). Antiviral treatment for Bell's palsy. *Cochrane Database Syst Rev*, 11, CD001869.
11. Hato, N., Yamada, H., Kohno, H., Matsumoto, S., Honda, N., Gyo, K., ... & Yamakawa, K. (2007). Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. *Otology & neurotology, 28*(3), 408-413.
12. Murthy, J. M., & Saxena, A. B. (2011). Bell's palsy: Treatment guidelines. *Ann Indian AcadNeurol, 14*(Suppl 1), S70-S73.
13. Teixeira, L. J., Soares, B. G., Vieira, V. P., & Prado, G. F. (2011). Physical therapy for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*, (12), CD006283.
14. Hesselink, J. R., & Quint, D. J. (2008). Neuroimaging in facial nerve disorders. *Neuroimaging Clin N Am, 18*(2), 341-359.